Migration Letters

Volume: 19, No: S5 (2022), pp. 1588-1592

ISSN: 1741-8984 (Print) ISSN: 1741-8992 (Online)

www.migrationletters.com

Nutritional Interventions For Chronic Disease Prevention And Management: A Critical Review

Bandar Mohammed Alhozim , Jaber Mohammed Ali Mobarki , Ali Hamad Hassan Qaysi, Sami Abdullah Ahmed Bajawi , Eman Mohammed Tokar Falatah , Abdulkarim Hadi Othman Hakmi , Mesfer Hamed Safar Almutairi , Naif Enad Motlaq Alotaibi , Khalid Hamad Ali Sahli , Moahmmed Bader Alotaibi

Abstract

The burden of noncommunicable illness may be tackled by using advancements in nutritional science, genetics, computer science, and behavioral economics. This research emphasizes the connection between nutrition and the intricate field of illness prevention and explores the advocacy of optimum metabolic health, drawing upon insights provided by other complementary fields. The discussion centers around two main topics. Firstly, it delves into the fundamental scientific aspects of achieving optimal metabolic health. The objective is to establish more precise targets and interventions. Secondly, it explores how nutrition, ranging from pharmaceutical approaches to lifestyle changes, can utilize systems science to tackle intricate problems.

Keywords: *gene-diet interactions, body weight, gut microbiota, systems science, diabetes, obesity.*

1. Introduction

The area of nutrition science is intricate as several components interplay to provide a desired result.1 The potency of a nutrient is influenced by various factors. These include the genetic makeup, age, and health status of the host. Additionally, the bioavailability of the nutrient is affected by conditions related to the growth, harvesting, storage, and preparation of food.2 Furthermore, the timing, frequency, and duration of dose, as well as contextual factors such as the presence of certain foods, medications, and diseases, can also impact nutrient absorption. While traditional linear, reductionist methods have been valuable in advancing our knowledge of nutritional factors and their impact on different diseases, they have limitations when it comes to studying intricate connections between variables such as dietary intake, activity levels, and disease manifestation. 3

Systems-based approaches facilitate the examination of the intricate manner in which nutrition interacts with genetic and environmental factors, thereby affecting the susceptibility to chronic diseases like obesity and type 2 diabetes. These approaches integrate data from multiple complementary disciplines to elucidate the progression of these diseases. 4, 5, 6. A conference titled "Nutrition and the science of disease prevention: a systems approach to support metabolic health" was organized by the Sackler Institute for Nutrition Science at the New York Academy of Sciences on April 16, 2015. The conference brought together prominent researchers in genetics, physiology, microbiology, epidemiology, and behavioral sciences to discuss the application of systems-based approaches in studying nutrition and metabolic health. This report offers a summary of the subjects discussed at the conference, with a particular emphasis on two main areas: (1) the fundamental scientific understanding of achieving optimal metabolic health, incorporating findings from studies on gene-diet interactions, the microbiome, and epidemiological research in nutrition, with the aim of identifying more effective targets and interventions, and (2) the ways in which nutrition, ranging from pharmaceutical approaches to lifestyle changes, can utilize systems science to tackle intricate problems.

2. The fundamental principles of achieving excellent metabolic health

Rudolph Leibel from Columbia University started the first session by delivering a talk on the fundamental molecular mechanisms behind body weight control, as well as more recent approaches for comprehending this process. From an evolutionary standpoint, body fat plays a crucial role in maintaining reproductive health and ensuring survival in situations when food is limited and sporadic. Longitudinal studies consistently demonstrate that people who achieve significant weight reduction typically have subsequent weight regain, resulting in a return to their initial weight. 7, 8, 9. While there are a few people who are able to successfully sustain weight loss over a long period of time, this is only possible with meticulous control of eating habits and physical activity.8

The phenomenon of weight regain after weight reduction can be attributed to the compensatory mechanisms of metabolic, behavioral, neuroendocrine, and autonomic systems that aim to preserve the body's energy (fat) reserves. These systems respond more strongly to weight loss than weight gain, exhibiting homeostatic reactions to changes in body fat.10 These mechanisms regulate body weight by maintaining it around a certain point called the "set point."

However, it is important to note that the regulatory factors are not balanced and instead favor the defense of body fat. Therefore, Leibel prefers to use the term "threshold" instead of "set point." Leptin, a hormone released by fat cells, plays a significant role in this system. It is produced in relation to the size and quantity of fat cells, meaning that people who are obese (with more and bigger fat cells) will generate higher levels of leptin compared to lean persons. Leptin passes over the barrier between the blood and the brain, mainly affecting two specific areas of the brain: the arcuate nucleus of the hypothalamus and the brainstem. The primary role of leptin in these locations is to communicate when the body is in a condition of low energy and reduced energy reserves.

Both obese and lean individuals experience a significant increase in energy intake and a decrease in energy expenditure when their leptin levels fall below a specific threshold. This response, known as an anabolic response, is triggered by leptin and aims to restore the individual's original body weight. It is characterized by delayed satiation and a perception of consuming fewer calories.12 The threshold for determining whether a person is lean or obese varies depending on genetic variables (such as MC4R and FTO genes), developmental influences (including pre- and postnatal impacts on brain development), and environmental factors. Obese persons have greater levels of leptin at which their energy expenditure reduces, compared to thin ones. Weight loss, on the other hand, does not change this limit, which implies that the systems governed by leptin in persons who have lost weight will still detect a lack of body fat. This lack is communicated physiologically in two ways: an increase in hunger and a decrease in energy expenditure, both of which promote weight return.10

Leibel reported findings from a set of weight-perturbation tests, where human participants were examined at their starting weight and after losing 10% of their body weight. Leptin was supplied at dosages that restored leptin levels to their original levels before weight loss, thus indicating to the brain that fat loss had not occurred. Liebel and colleagues discovered that administering leptin to persons who had lost weight led to a lesser decrease in energy expenditure, enhanced satiation, and reversed the typical rise in muscle contractile efficiency that happens with weight loss. 13, 14. Furthermore, neuroimaging studies have shown that the injection of leptin to individuals who have lost weight restores activity in many parts of the brain that are sensitive to leptin, such as the hypothalamus, which are often affected after weight loss.15

Liebel then proceeded to analyze the consequences of these discoveries for the treatment of obesity. Research has shown that the major difficulty faced by most people is not in shedding pounds, but in sustaining weight loss in the long term. Moreover, from a physiological or pharmacological standpoint, it is easier to restore normal bodily functions than it is to initiate weight loss. Liebel proposed that the primary objective of obesity treatment should be to restore normal physiological functions in those who have lost weight, rather than only focusing on weight reduction. As previously said, a method to return the body to its normal functioning is to intentionally communicate to the brain that weight loss has not occurred. This may be achieved by increasing the levels of leptin after reducing weight.

3. Nutrition and the science of disease prevention

Researchers discussed advanced systems science techniques that allow for the examination of intricate issues. These methods have the potential to enhance nutrition and obesity research by providing alternative approaches to conventional methodologies. Mabry provided a comprehensive analysis of systems science, highlighting its ability to study complex problems in a manageable way by simplifying the problem while preserving its important features. Additionally, systems science examines both the overall structure of a complex problem and its individual elements. Systems science approaches are specifically developed to capture the dynamic behavior of a system over time. They are used to examine the two-way interactions and non-linear connections within the system, as well as the impacts that are delayed in time, such as the impact of nutrition-related regulations on the intake of impacted foods.

In addition, the application of systems science methods can facilitate the identification of unintended consequences, such as the situation where the implementation of taxes on sugar-sweetened beverages results in consumers avoiding these beverages but increasing their consumption of artificially sweetened beverages that are not subject to taxation. Furthermore, these methods can also help uncover emergent properties, where the collective behavior of individuals leads to a larger-scale outcome. These methods are especially useful for conducting virtual experimentation, such as in in silico laboratories. This type of experimentation is based on simplified data within a simulation model and allows for the assessment of the effects of nutrition-related policies without having to wait for decades to see the full results.

Modeling and simulation are prevalent in the majority of systems science approaches. Mabry examined the significance and practicality of modeling, including their role in making mental models clear and explicit; elucidating (rather than forecasting); guiding data collection (e.g., determining the necessary data to enhance the model's strength and dependability); revealing fundamental dynamics; illustrating trade-offs; and bounding outcomes with credible ranges (Mabry emphasized that while models may not excel at precise predictions, they are valuable in establishing plausible ranges within which outcomes may occur).

Mabry presented the Prevention Impacts Simulation Model (PRISM) as an illustration of how system dynamics modeling can be used to provide information for making food policy decisions at the community level. PRISM is a thorough and evidence-based simulator that operates on the principles of system dynamics. It is developed by the Centers for Disease Control (CDC) with assistance from the National Heart, Lung, and Blood Institute (NHLBI) and the National Institutes of Health (NIH) Office of Behavioral and Social Sciences Research (OBSSR). The primary purpose of PRISM was to analyze how different policies might be integrated to effectively decrease mortality and costs associated with cardiovascular disease (CVD) and other chronic diseases.

PRISM incorporates the most reliable evidence into a framework that can be tested and used for future planning and assessment. The model has the capability to integrate a minimum of 34 potential treatments, such as implementing a tax on junk food, introducing measures to promote fruit and vegetable consumption, reducing salt and trans fats, and implementing physical activity interventions. It assesses the effectiveness of these interventions and their impact on the desired result, such as cardiovascular disease, over a period of time. For instance, several studies utilizing PRISM have analyzed the impact of established risk factors, like physical activity, on obesity rates at different time intervals to evaluate if physical activity continues to have a lasting effect. Additionally, these studies have also forecasted the consequences of interventions that target obesity, such as the implementation of a tax on junk food.16-20

Furthermore, alongside system dynamics modeling, Mabry also presented agent-based modeling as a method for doing obesity research. Specifically, she emphasized a recent model created by Hammond et al.21 that aims to investigate the interplay between genetic (dopamine), environmental (food geography, economic factors), social (body image, eating norms), physiological (energy regulation), and neurobiological (reward learning, executive control, homeostasis) factors. The objective is to understand how these factors collectively impact eating behavior, body weight, and obesity. 22,23 Mabry concluded her presentation by offering details on diverse resources in systems science applications in public health and nutrition research,5,24 as well as an overview of systems science-related initiatives at the NIH and federal funding prospects for research utilizing systems science-based methodologies. 25,26

4. Summary

The relationship between diet and the development and prevention of metabolic illnesses is intricate. Systems-level methodologies may include diverse data from complementary fields to elucidate the biological processes that underlie metabolic illness and to forecast the impact of nutrition-related policy measures on disease outcomes. Applying systems-level approaches to nutrition research has revealed the intricate connections between nutrition and various factors such as neurobiology, endocrinology, genetics, gut microbiota, behavior, and the environment. These connections play a role in metabolic health and the onset of chronic diseases like obesity and type-2 diabetes. Systems approaches, such as those used in epidemiology, can provide a comprehensive understanding of the complex connections between dietary factors and metabolic diseases. By delving into the molecular and behavioral pathways involved, these approaches have the potential to reveal the underlying mechanisms behind the observed relationships in epidemiological and clinical studies, thus shedding light on the "black box" of chronic disease epidemiology. Research using systems techniques may enhance the early identification, clinical diagnosis, and prognosis of diseases. It can also aid in developing individualized dietary therapies to promote metabolic health and provide valuable insights for policy-level interventions.

References

1. Mocchegiani E, Costarelli L, Giacconi R, et al. Micronutrient-gene interactions related to inflammatory/immune response and antioxidant activity in ageing and inflammation. A systematic review. Mech Ageing Dev. 2014;136–137:29–49.
2. Amarowicz R, Carle R, Dongowski G, et al. Influence of postharvest processing and storage on the content of phenolic acids and flavonoids in foods. Mol Nutr Food Res. 2009;53:S151–S183
3. Genser D. Food and drug interaction: consequences for the nutrition/health status. Ann Nutr Metab. 2008;52:29–32.

4. Franks PW, Pearson E, Florez JC. Gene-environment and gene-treatment interactions in type 2 diabetes: progress, pitfalls, and prospects. Diabetes Care. 2013;36:1413–1421.
5. Mabry PL, Bures RM. Systems science for obesity-related research questions: an introduction to the theme issue. Am J Public Health. 2014;104:1157–1159.
6. Astarita G, Langridge J. An emerging role for metabolomics in nutrition science. J Nutrigenet Nutrigenomics. 2013;6:181–200.
7. Atkinson RL, et al. Very low-calorie diets. National Task Force on the Prevention and Treatment of Obesity, National Institutes of Health. JAMA. 1993;270:967–974.
8. Look AHEAD Research Group et al. Reduction in weight and cardiovascular disease risk factors in individuals with type 2 diabetes: one-year results of the look AHEAD trial. Diabetes Care. 2007;30:1374–1383.
9. Foster GD, et al. Weight and metabolic outcomes after 2 years on a low-carbohydrate versus low-fat diet: a randomized trial. Ann Intern Med. 2010;153:147–157.
10. Leibel RL. Molecular physiology of weight regulation in mice and humans. Int J Obes (Lond) 2008;32:S98–S108.
11. Rosenbaum M, Leibel RL. 20 years of leptin: role of leptin in energy homeostasis in humans. J Endocrinol. 2014;223:T83–T96.
12. Rosenbaum M, Leibel RL. Adaptive thermogenesis in humans. Int J Obes (Lond) 2010;34:S47–S55.
13. Leibel R, Rosenbaum M, Hirsch J, et al. Changes in energy expenditure resulting from altered body weight. N Engl J Med. 1995;332:621–628.
14. Kissileff HR, Thornton JC, Torres MI, et al. Leptin reverses declines in satiation in weight-reduced obese humans. Am J Clin Nutr. 2012;95:309–317.
15. Begg DP, Woods SC. The endocrinology of food intake. Nat Rev Endocrinol. 2013;9:584–597
16. Homer J, Milstein B, Wile K, Pratibhu P, Farris R, Orenstein DR. Modeling the local dynamics of cardiovascular health: risk factors, context, and capacity. Prev Chronic Dis. 2008;5:A63.
17. Homer J, Milstein B, Wile K, Trogdon J, Huang P, Labarthe D, et al. Simulating and evaluating local interventions to improve cardiovascular health. Prev Chronic Dis. 2010;7(1):1–11
18. Homer J, Wile K, Yarnoff B, Trogdon JG, Hirsch G, Cooper L, Soler R, Orenstein D. Using Simulation to Compare Established and Emerging Interventions to Reduce Cardiovascular Disease Risk in the United States. Prev Chronic Dis. 2014;11:E195.
19. Loyo HK, Batcher C, Wile K, Huang P, Orenstein D, Milstein B. From model to action: using a system dynamics model of chronic disease risks to align community action. Health Promotion Practice. 2013;14(1):53–61.
20. Honeycutt AA, Wile K, Dove C, Hawkins J, Orenstein D. Strategic Planning for Chronic Disease Prevention in Rural America: Looking Through a PRISM Lens. Journal of Public Health Management and Practice: (JPHMP) 2015;21:392–399.
21. Hammond RA, Ornstein JT, Fellows LK, Dube L, Levitan R, Dagher A. A Model of Food Reward Learning with Dynamic Reward Exposure. Frontiers in Computational Neuroscience. 2012;6(82):1–8.
22. Hall KD, Hammond RA, Rahmandad H. Dynamic interplay among homeostatic, hedonic, and cognitive feedback circuits regulating body weight. American Journal of Public Health. 2014;104(7):1169–1175.
23. Shoham DA, Hammond R, Rahmandad H, Wang Y, Hovmand P. Modeling Social Norms and Social Influence in Obesity. Current Epidemiology Reports. 2015:1–9.
24. Mabry PL, Milstein B. Supplemental issue: systems science applications in health promotion and public health. Health Educ Behav. 2013;40:5S–131S.
25. National Institutes of Health. Systems science and health in the behavioral and social sciences (R01) Accessed August 12, 2015 http://grants.nih.gov/grants/guide/pa-files/PAR-15-048.html.
26. National Institutes of Health. Systems science and Health in the Behavioral and Social Sciences (R21) Accessed August 12, 2015 http://grants.nih.gov/grants/guide/pa-files/PAR-15-047.html.